IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Appl. No. : 10/511,627
Applicant : Karsten Eulenberg et al
Filed : October 18, 2004
TC/A.U. : 1656
Examiner : Rita Mitra

Docket No. : 2923-657
Customer No. : 6449
Confirmation No.: 8622

DECLARATION UNDER 37 C.F.R. 1.132

Director of the United States Patent
and Trademark Office
P.O. Box 1450
Alexandria, Virginia 22313-1450

DRAFT

I, Dr. Marcus Geese, hereby declare as follows:

1. Experiments were carried out under my direction and control which show the effects of RNAi mediated loss of function in 3T3-L1 adipocytes. These experiments show that

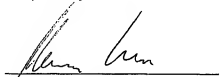
- a knockout of SAC2 by an RNAi molecule leads to a decreased expression of adipocyte markers such as PPARgamma (Figure 1) and aP2 (Figure 2) throughout adipogenesis,
- a SAC2 knockout results in a decreased triglyceride content (Figure 3) through adipogenesis,
- a SAC2 knockout reduces the free fatty acid uptake in adipocytes (Figure 4) and
- a knockout of SAC2 leads to an increase of insulin-stimulated glucose uptake (Figure 5).

The experiments were conducted as shown in the attached document entitled "CG7956 Homolog in Vitro Validation".

2. One skilled in the art would reasonably conclude from these experiments that administering a modulator (in particular. an inhibitor) of CG7956 nucleic acid molecule, a modulator (in particular. an inhibitor) of a polypeptide encoded thereby according to SEQ ID NO:14, SEQ ID NO:15 or ENSMUSP00000045910, or a modulator (in particular. an inhibitor) of a fusion polypeptide comprising said polypeptide, to a patient in need of such treatment would be effective to treat metabolic diseases or dysfunctions, and that a modulator (in particular. an inhibitor) of CG7956 nucleic acid molecule, a modulator (in particular. an inhibitor) of polypeptide encoded thereby according to SEQ ID NO:14, SEQ ID NO:15 or ENSMUSP00000045910, or a modulator (in particular. an inhibitor) of a fusion polypeptide comprising said polypeptide, can be used to regulate triglyceride metabolism and/or adipogenesis.

3. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Respectfully submitted,

By: 

Dr. Marcus Geese

Date: 2007-08-14

Marcus Geese, Ph. D.

Wilhelm-Weber-Str. 35
37073 Gottingen
Germany

phone: +49-551-50558-620
e-mail: geese@develogen.com

Marcus Geese

Curriculum Vitae

August 14th, 2007

Employment History

DeveloGen AG, Germany

since 05/2006	Associate Director In Vitro Pharmacology
07/2004 - 05/2006	Senior Scientist In Vitro Pharmacology
05/2002 - 06/2004	Scientist In Vitro Pharmacology

GBF - German Research Centre for Biotechnology, Germany

12/2001 - 04/2002	Postdoc Research

Scientific Education

GBF - German Research Centre for Biotechnology, Germany

08/1998 - 11/2001	Ph.D. ("magna cum laude")
10/1993 - 07/1998	Diploma in Biology ("Diplom-Biologe")

Publications

- Mussmann R, Geese M, Harder F, Kegel S, Andag U, Lomow A, Burk U, Onichtchouk D, Dohrmann C, Austen M.: Inhibition of GSK3 Promotes Replication and Survival of Pancreatic Beta Cells. J Biol Chem. 2007;282(16): 12030-7
- Grenklo S, Geese M, Lindberg U, Wehland J, Karlsson R, Sechi AS: A crucial role for profilin-actin in the intracellular motility of Listeria monocytogenes. EMBO Rep. 2003; 4(5):523-9
- Geese M, Loureiro JJ, Bear JE, Wehland J, Gertler FB, Sechi AS: Contribution of Ena/VASP proteins to intracellular motility of Listeria requires phosphorylation and proline-rich core but not F-actin binding or multimerization. Mol. Biol. Cell. 2002; 13: 2383-96
- Geese M, Schlüter K, Rothkegel M, Jockusch BM, Wehland J, Sechi AS: Accumulation of profilin II at the surface of Listeria is concomitant with the onset of motility and correlates with bacterial speed. J. Cell. Sci. 2000; 113: 1415-1426